Dietary Fiber and Prevention of Cardiovascular Disease*

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In 1867, the great American artist Winslow Homer created a woodcarving depicting a man standing alone, wielding a scythe against an endless field of wheat as tall as the man himself. The work is entitled *The Veteran in a New Field*. This image comes to mind when I contemplate the history of dietary fiber and disease prevention. The idea that dietary fiber from foods like whole grains may have health-promoting effects has been around a long time. Yet even in the age of genomics, proteomics, high-tech imaging and the emphasis on discovery of “new” novel risk factors for heart disease, we return to the role of dietary fiber, and it remains keenly relevant today.

In 1971, British surgeon Denis Burkitt revitalized interest in dietary fiber by providing epidemiologic evidence that it may be protective against disease [1]. Not only were his observations—that the high-residue content of the diet of black African populations compared to white African populations consuming a “Western”-type diet—consistent with a protective effect against colon cancer, but also his work stimulated research into the role of dietary fiber in the etiology of other diseases, including cardiovascular disease.

By 1982, Kromhout et al. [2] had demonstrated a statistically significant reduction in coronary heart disease associated with an increase in dietary fiber in the 10-year follow-up of the Zutphen study. Since then, several investigators have reported modest but consistent reductions in risk of coronary heart disease associated with increased dietary fiber. The article from the Woman’s Health Study by Liu et al. [3] in this issue of the *Journal* is the latest prospective study to report on the risk of cardiovascular disease associated with fiber intake.

Three decades after Burkitt’s observations, defining the role of dietary fiber in coronary heart disease risk is still vitally important for three reasons. First, an understanding of the role of fiber in coronary heart disease risk can help clarify and reinforce dietary recommendations and goals. This is particularly important because the public is repeatedly exposed to conflicting and often spurious claims about the effectiveness of dietary components in preventing a variety of conditions. We need clear messages about the role and magnitude of the effect of dietary fiber that are firmly based on good science.

Second, the mechanisms of how dietary fiber may reduce cardiovascular disease risk are not yet fully understood. It appears that the protective effect of dietary fiber is independent of its effect on serum cholesterol levels. The cholesterol-lowering effect of dietary fiber has been shown to be modest at best [4], and the reduced risk of coronary heart disease associated with whole grain intake may be only partly due to its contribution to total fiber intake [5]. Insights into the mechanisms of fiber’s protective effect may lead to better dietary recommendations and new prevention strategies.

Third, increasing the fiber content in the diet is relatively low-cost, safe and associated with few side effects, making it a viable option for a large proportion of the population. If its effect is truly protective, even if only modestly so, it has the potential to have a large impact on disease rates at the population level because the prevalence of low-fiber diets in the U.S. is substantial. Of course, increasing the knowledge about dietary fiber can be beneficial. But even if the mechanisms are clearly identified and the estimates of risk reduction are valid, there is no guarantee that the behavioral changes in the population will occur.

Although the recent findings by Liu et al. [3] largely agree with the existing literature, this article makes several important contributions to the body of evidence on the role of dietary fiber in preventing cardiovascular disease. One contribution is that the observational design assesses the effect of an amount of dietary fiber intake that is practicable, albeit still below recommended levels. Dietary guidelines from the American Heart Association, though not recommending a specific amount of dietary fiber, state that the consumption of the amounts and types of foods in their recommendations should translate to >25 g of fiber per day [6]. This is about twice the current national average of 14 to 15 g today in the U.S. [7]. The intake among professional women in the Women’s Health Study reported by Liu et al. [3] was between the national average and recommended levels. The median dietary fiber intake in the lowest quintile in the Women’s Health Study was 18 g/d, and the median for the highest quintile was 26 g/d. A recent metabolic study of 10 healthy volunteers demonstrated that very high-fiber diets (55 g/1,000 kcal) resulted in a 33% reduction in low-density lipoprotein cholesterol over a two-week period [8]. This level of fiber intake is 3.6 times that of the median of the highest quintile of intake in the study by Liu et al. [3] and is not practical on a population basis. Therefore, the modest effects on cardiovascular disease risk observed in the study by Liu et al. [3] are in response to what could be considered a modest yet feasible level of habitual intake.

Another contribution of the article by Liu et al. [3] is that they present data on both a combined end point of cardio-

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vascular disease (defined as nonfatal myocardial infarction [MI], revascularization procedure or stroke) as well as for MI only. In general, the trends seen for the combined end point agree with the more specific MI end point, with the latter showing slightly stronger associations. This is important because it provides insight into the specificity of effect within the same study population. The weakening of the effect when considering the combined end point may be due to the inclusion of stroke events. However, this is unlikely because there is evidence of an inverse association between fiber intake and stroke risk (9). Two thirds of the combined end point were not MI. Therefore, it is expected that this would add some heterogeneity to the outcome measure and weaken the strength of association. What is not known from the data presented by Liu et al. (3) is whether or not factors affecting access to revascularization procedures, which likely make up the majority of the non-MI end points, are also related to dietary fiber intake among this study population.

The article by Liu et al. (3) also contributes valuable data on fiber source and type, which could provide insights into possible mechanisms. The small differences they found in the effect of soluble versus insoluble fiber, however, were not meaningful. Neither soluble nor insoluble fiber was statistically significantly related to cardiovascular disease or MI in multivariate models. Similar null findings were seen when the analysis was stratified on cereal versus vegetable fiber.

Although this article contributes >230,000 person years of careful and considered observation, there are some relevant limitations of the study. One is the limited absolute range of fiber intake among this cohort. The difference in median intake from lowest quintile to highest quintile is only 8.1 g of fiber per day. This is less than one serving of a high-fiber cereal or the combined fiber in a serving of peas, a potato and an apple. This limited range of exposure decreased their ability to show a dose/response relationship between fiber and cardiovascular disease risk. It is possible that the amount of fiber consumed is below a threshold at which benefit is most evident. Without data on a wider range of fiber intake, it is difficult to characterize the potential benefit of the upper range of current dietary recommendations. Without additional data, it is difficult to determine whether or not the risk estimates observed by Liu et al. (3) are, in fact, underestimates. Furthermore, a single measure of diet is not likely to fully characterize the influence of fiber over a person's lifetime.

The central questions at hand are these: Is the association between dietary fiber and cardiovascular disease valid, and is it causal? Relevant to the first count of validity, Liu et al. (3) address the role of chance, bias and confounding. The 95% confidence interval (CI) around the age, treatment-adjusted relative risk (RR) for cardiovascular disease (RR = 0.65, 0.51 to 0.84) and MI (RR = 0.46, 0.30 to 0.72), suggest that these associations were not observed by chance. Bias was also addressed in this study. The prospective design likely reduced the role bias in that diet assessment was determined prior to occurrence of the primary outcome. The role of confounding, however, is less easily dismissed. All but 2 of the 12 multivariate-adjusted models reported among the full cohort attenuated the RR from the age treatment-adjusted models and rendered them nonstatistically significant. The two that did not were already nonsignificant in the more basic model and remained so in the multivariate model. This suggests that a substantial portion of the relationship between dietary fiber and cardiovascular disease risk reduction was due to confounding by other factors. Under a strict interpretation of the data, one cannot rule out the assertion that in this study, there was no independent effect of dietary fiber on cardiovascular disease risk. However, in two subgroup analyses, the CIs around the multivariate adjusted RR did not include the null value (those whose body mass index was <25 and among those who never smoked).

The second count of causality requires a more global look at the evidence and application of the scientific method to available epidemiologic data, then a repetition of the process when new data come along. The study by Liu et al. (3) does not answer the question of causality—indeed, no single observational study can. However, their data are consistent with a growing body of evidence in support of current dietary recommendations to incorporate more fiber from whole grains and fruits and vegetables into the diet for the prevention of heart disease. Liu et al. (3) put their work in context nicely by summarizing 10 previous prospective studies. Their pooled analysis involving 10 published prospective studies produced an RR of coronary heart disease of 0.83 (95% CI 0.78 to 0.89) associated with a 10-g increase in dietary fiber intake. Although this is reassuring, more needs to be done to translate epidemiologic evidence and recommendations into effective practice. This takes us back to Homer’s wheat field. We have learned much about dietary fiber and cardiovascular disease, but there is still much left to harvest.

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